



NOTES

ERYTHEMA MULTIFORME & DRUG ERUPTION

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Skin, mucous membrane conditions
- Associated with medication use/infection

CAUSES

- Exact mechanism unclear, severe immune reaction against foreign antigen

COMPLICATIONS

- Initial rash may → epidermal layer loss

SIGNS & SYMPTOMS

- Desquamating skin, mucous membrane rash

DIAGNOSIS

LAB RESULTS

- Skin biopsy

OTHER DIAGNOSTICS

- Clinical history

TREATMENT

- Identify/remove/treat offending agent/infection

ERYTHEMA MULTIFORME

osms.it/erythema-multiforme

PATHOLOGY & CAUSES

- Immune-mediated, acute, self-limiting skin condition
- Type IV hypersensitivity

CAUSES

- Suspected deposition of primarily IgM-bound immune complexes in superficial skin, oral mucous membranes

Infection (most)

- Viral
 - Herpes simplex primary cause

- Bacterial
 - Hemolytic Streptococci, Legionella, Mycobacterium, *Mycoplasma pneumoniae*, Neisseria meningitidis, Pneumococcus, Salmonella, Staphylococcus
- Parasitic
 - Trichomonas, Toxoplasma gondii

Drugs (rarely)

- Non-steroidal anti-inflammatories (NSAIDs), sulfonamides, phenytoin, barbiturates, phenylbutazone, penicillin, allopurinol

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Physical factors

- Sunlight, radiotherapy, cold

Autoimmune disease

- Vasculitides

Hematological malignancy

- Non-Hodgkin lymphoma, leukemia, myeloid metaplasia

RISK FACTORS

- < 20 years old
- ↑ frequency in biological males

SIGNS & SYMPTOMS

- “Multiforme” denotes **wide associated lesion variety**
- **Target lesions**
 - Initially round erythematous papules → dusky central area/blister, surrounded by dark red inflammation, surrounded by pale edematous ring, erythematous region on periphery
- Pruritus in affected area
- Painful lesions
- If severe
 - Fever, weakness, malaise



Figure 3.1 The abdomen of a child displaying numerous target lesions in a case of erythema multiforme.

Erythema multiforme minor

- Often herpes simplex
- Involves skin (little/no mucous membrane involvement)
- Favors skin of extremities, face
- Symmetrical circular lesions
- Lesions become classic “target” lesions (red border, small white center)
- Rash spreads towards body center

Erythema multiforme major

- Often drug-related
- Epidermal detachment/skin loss progression
- Erythematous, confluent, bullous lesions
- Involves mucous membranes
- Nikolsky’s sign (lightly rub skin with firm object for few seconds → blister forms)

DIAGNOSIS**LAB RESULTS**

- Biopsy to exclude other skin disorders

OTHER DIAGNOSTICS

- Identify offending agent/infection
 - **Identification:** target lesions, symmetrical distribution

TREATMENT

- Often self-resolving in 1–2 weeks

MEDICATIONS

- Control primary cause
 - Treat/remove identifiable causes
 - **Herpes simplex suspected:** oral acyclovir/valaciclovir/famciclovir
 - Eliminate possible offending drugs

Mild disease

- Topical corticosteroids
- Antihistamines

Severe Disease

- Glucocorticoids
- In severe cases, prednisone considered

Recurrent disease

- Systemic antivirals (up to 6 months)
- Immunosuppression if antivirals fail

STEVENS–JOHNSON SYNDROME & TOXIC EPIDERMAL NECROLYSIS

osms.it/stevens-johnson_syndrome

osms.it/toxic-epidermal-necrolysis

PATHOLOGY & CAUSES

- Same underlying pathology (severity spectrum)
 - Stevens–Johnson syndrome (lower end), toxic epidermal necrolysis (upper end)
- Severity, classification
 - Body surface involvement %
- Severe mucocutaneous reaction → epidermal detachment

CAUSES

- Cytotoxic T cell mediated destruction of keratinocytes expressing foreign antigen

Medications

- Most common
- Allopurinol, sulfa drugs (e.g. sulfonamide antibiotics), lamotrigine, carbamazepine, nevirapine, phenylbutazone, thiacetazone, oxicam NSAIDS

Infections

- Mycoplasma pneumoniae most common infective agent

RISK FACTORS

- HIV/AIDS
- Systemic lupus erythematosus
- > 40 years old
- Genetic carbamazepine interaction predisposition (HLA-B*15:02, HLA-A*31:01 alleles)
- ↑ frequency in biological females

COMPLICATIONS

- Dehydration, sepsis, pneumonia, multiple organ failure, renal tubular necrosis, acute renal failure, phimosis, vaginal synechiae (adhesions), inside eyelid-tissue scarring → corneal vascularisation → vision loss



Figure 3.2 An individual with Stevens–Johnson syndrome.

SIGNS & SYMPTOMS

Systemic

- Before skin eruptions occur
- Fever, sore throat, fatigue, cough

Mucocutaneous

- Burning eyes, skin
- Red-purple macules → skin blisters → peels, forms painful raw areas
- Mucous membranes (often) → painful crusts, erosions
- Starts on trunk → rest of body
- Spontaneous ulceration of skin, mucous membranes (often eyes/lips)
- Conjunctivitis (often accompanied by purulent discharge)
- Round ulcerating lesions (approx. 2.5cm/1in diameter)
 - Arise on face, trunk, arms, legs, soles of feet (scalp spared)
- Nikolsky's sign



Figure 3.3 An individual with toxic epidermal necrolysis ten days after the onset of symptoms.

DIAGNOSIS

- Stevens–Johnson syndrome: < 10% skin involvement
- SJS/TEN overlap: 10–30%
- Toxic epidermal necrolysis: > 30%

LAB RESULTS

- Skin biopsy

OTHER DIAGNOSTICS

- Clinical history, suspected agents

TREATMENT

MEDICATIONS

- Analgesics (non-opioid for non-severe, opioids for severe pain)
- Antihistamines
- Intravenous immunoglobulin

Infection control

- Culture-specific antibiotic initiation

OTHER INTERVENTIONS

- Transfer to burn/intensive care unit
- Fluid support
- Oral feeding, nasogastric tube
- Room temperature 30–32°C/86–90°F (minimize heat loss)

Infection control

- Sterile handling
- Skin disinfection
 - Antiseptic solution
- 48 hourly skin, blood, indwelling line culture

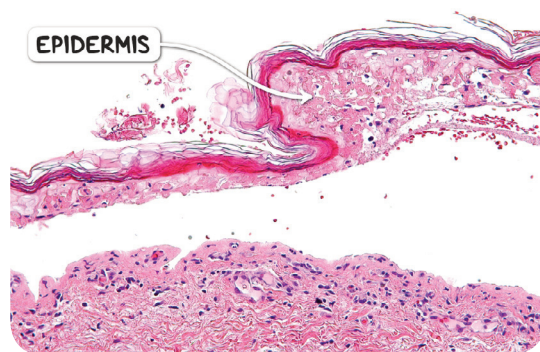


Figure 3.4 A histological section of skin demonstrating epidermal necrolysis. The epidermis is detached from the dermis and the keratinocytes have undergone necrosis. This can be seen in erythema multiforma, Stevens-Johnson syndrome and toxic epidermal necrolysis.